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FILE 'HOME' ENTERED AT 14:48:11 ON 08 AUG 2001

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FILE 'USPATFULL' ENTERED AT 14:48:23 ON 08 AUG 2001

=> s fusion (p) protein (p) steroid (p) receptor (p) thyroid

L1 69 FUSION (P) PROTEIN (P) STEROID (P) RECEPTOR (P) THYROID

=> s fusion (s) protein (s) steroid (s) receptor (s) thyroid

4 FILES SEARCHED...
L2 51 FUSION (S) PROTEIN (S) STEROID (S) RECEPTOR (S) THYROID

=> s fusion (s) protein (s) steroid (s) receptor (s) thyroid (s) hormone

4 FILES SEARCHED...
L3 35 FUSION (S) PROTEIN (S) STEROID (S) RECEPTOR (S) THYROID (S) HORMONE

=> dup rem l3

PROCESSING COMPLETED FOR L3
L4 16 DUP REM L3 (19 DUPLICATES REMOVED)

=> d l4 total ibib kwic

L4 ANSWER 1 OF 16 USPATFULL
ACCESSION NUMBER: 2000:9723 USPATFULL
TITLE: Unique nucleotide and amino acid sequence and uses thereof
INVENTOR(S): Summers, Max D., Bryan, TX, United States
Braunagel, Sharon C., Bryan, TX, United States
Hong, Tao, Bryan, TX, United States
PATENT ASSIGNEE(S): The Texas A & M University System, College Station, TX,

United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6017734		20000125
APPLICATION INFO.:	US 1997-792832		19970130 (8)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1996-678435, filed on 3 Jul 1996, now abandoned

	NUMBER	DATE
PRIORITY INFORMATION:	US 1995-955	19950707 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Elliott, George C.
ASSISTANT EXAMINER: Schwartzman, Robert
LEGAL REPRESENTATIVE: Arnold, White & Durkee
NUMBER OF CLAIMS: 56
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 47 Drawing Figure(s); 24 Drawing Page(s)
LINE COUNT: 7846
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
DETD . . . Nakhai et al. 1991
choriogonadotropin .beta.-subunit Chen et al. 1991
choriogonadotropin .beta.-subunit Chen and Bahl 1991
descarboxyl-terminal peptide
chorionic gonadotropin **hormone** Nakhai et al. 1991
precursor
chorionic gonadotropin **hormone** Hasnain et al. 1994
(b-subunit)
chorionic gonadotropin **hormone** Nakhai et al. 1992
b subunit
complement Clr Sass et al. 19??
complement Clr proenzyme Gal et al. 1989
complement **protein** C9 Tomlinson et al. 1993
corticosteroid binding globulin Ghose Dastidar et al. 1991
c-myc **protein** Miyamoto et al. 1985
complement **protein** C9 Tomlinson et al. 1993
corticosteroid binding globulin Ghose-Dastidar et al. 1991
(hCBG)
creatine kinase B (B-CK) de Kok et. . . 1994
phosphoprotein
cytomegalovirus IE1, IE1 exon 4 Davrinche et al. 1993
cytosolic phospholipase A.sub.2 Abdullah et al. 1995
D4 dopamine **receptor** Mills et al. 1993
DNA ligase I Gallina et al. 1995
DNA polymerase a subunit Copeland and Wang 1991
DNA polymerase d catalytic subunit Zhou et al. 1996
DNA topoisomerase 1 Zhelkovsky & Moore 1994
dopamine D.sub.2 **receptor** Javitch et al. 1994
EGF **receptor** Greenfield et al. 1988
EGF **receptor**-tyrosine kinase domain Wedegaertner et al. 1989
endothelial nitric-oide synthase Chen et al. 1996
epidermal growth factor **receptor** Waterfield & Greenfield 1991
epidermal-growth-factor **receptor** McGlynn et al. 1992
protein-tyrosine kinase
epidermal growth factors IX and XIIa Astermark et al. 1994
erythrocyte anion exchanger Dale et al. 1996
erythropoietin Quelle et al. 1992
estrogen **receptor** Beekman et al. 1994
factor VIII - B domain deleted Webb et al. 1993
fibroblast growth factor **receptor** Sisk et al. 1992
subtype ligand binding domain
follicle-stimulating **hormone receptor** Christophe et al.
1993
furin Bravo et al. 1994

GABA.sub.A **receptor** a1 subunits Birnir et al. 1995
GABA.sub.A **receptor** b1 subunits Birnir et al. 1995
ga773 - 2 antigen Strassburg et al. 1992
GMP synthetase Lou et al. 1995
glucocerebrosidase Martin et al. 1988
glucocorticoid **receptor** Srinivasan et al. 1990
glutamic acid decarboxylase Mauch et al. 1993
glycine **receptor** a1 Morr et al. 1995
group b rotavirus ADRV, VP4 Mackow et al. 1993
group II Phospholipase A.sub.2 Tremblay et al. 1993
growth **hormone** Sumathy et al. 1996
growth **hormone receptor** - Ota et al. 1991
extracellular domain
5-HT.sub.1A **receptor** Mulheron et al. 1994
hst-1 transforming **protein** Miyagawa et al. 1988
heart (R)-3-hydroxybutyrate Green et al. 1996
dehydrogenase
hematopoietic glycopeptide Quelle et al. 1992
erythropoietin
hemopexin Satoh et al. 1994
heparin cofactor II Ciaccia et al. 1995
hepatitis b virus X **protein** Klein et al. 1992
hepatocyte growth factor Yee et al. 1993
hepatocyte growth factor Lee et al. 1993
high-affinity IgE **receptor**-a chain Yagi et al. 1994
17b-hydroxysterioid dehydrogenase Breton et al. 1994
5-hydroxytryptamine.sub.1A Butkerait et al. 1995
5-hydroxytryptamine **receptors** Parker et al. 1994
(5-HT.sub.1A, 5-HT.sub.1Da, 5-HT.sub.1Db, 5-HT.sub.1E)
IgA Carayannopoulos et al. 1994
IL2 **receptor** a & b chains Lindqvist et al. 1993
immunodeficiency virus-type 1 gag Chazal et al. 1994
precursor
immunodeficiency virus-1 gp41 Lu et al. 1993
immunodeficiency virus-1 gp120 Yeh et al. 1993
insulin holoreceptor Paul et al. 1990
insulin **receptor** substrate-1 Siemeister et al. 1995
insulin **receptor** b-subunit Herrera et al. 1988
insulin **receptor** b subunit Li et al. 1992
transmembrane/cytoplasmic domain
insulin **receptor** ectodomain Sissom et al. 1989; 1991
insulin **receptor protein**-tryosine Ellis et al. 1988
kinase domain
insulin **receptor** cytoplasmic domain Herrera et al. 1988
of b subunit
insulin **receptor protein** tyrosine- Ellis and Levine 1991
kinase-cytoplasmic domain
insulin-like growth factor II Congote and Li, 1994
insulin-like growth factor II Marumoto. . . glycoforms Ogonah et al. 1995
interleukin 2 Smith et al. 1985
interleukin 2 glycoprotein variants Grabenhorst et al. 1993
interleukin-2 **receptor** gamma chain Raivio et al. 1995
interleukin 5 Brown et al. 1995
interleukin 6 Matsuura et al. 1991
interleukin-6 **receptor** Weiergraber et al. 1995
intrinsic factor Gordon et al. 1992
iron regulatory factor Emery-Goodman et al. 1993
isoforms (neuronal, inducible,. . . Ku autoantigen Allaway et al. 1990
lecithin-cholesterol acyltransferase Chawla & Owen 1995
Leukotriene A.sub.4 hydrolase Gierse et al. 1993
link **protein** Grover & Roughley 1994
liver carboxylesterase Kroetz et al. 1993
lymphocytic activation gene (LAG-1) Baizleras et al. 1990
lysyl hydroxylase. . . al. 1996
lysosomal b-galactosidase Itoh et al. 1991
5'lipoxygenase Dunk et al. 1989

m1 muscarinic acetylcholine Haga et al. 1996
receptors
m2 muscarinic cholinergic **receptor** Debburman et al. 1995
m3 (hm3) muscarinic cholinergic Debburman et al. 1995
receptors
MHC class I HLA-b27 antigen Levy and Kvist 1990
MHC class II DR4a, DR4b, extra- Scheerle et al. 1992
cellular. . . colony stimulating factor Qiu et al. 1995
matrilysin Lopez de Turiso et al. 1996
metallothionein-II Schmiel et al. 1985
mineralocorticosteriod **receptor** Binart et al. 1991
monocyte chemoattractant **protein**-1 Ueda et al. 1994
Ishii et al. 1995
multidrug resistance 1 Germann et al. 1990
multidrug resistance P-glycoprotein Rao et al. 1994
muscarine **receptor** m2 Kameyama et al. 1994
myeloperoxidase Taylor et al. 1992
myogenic factors myf4, myf5 Braun et al. 1991
N-formyl peptide **receptor** Quehenberger et al. 1992
Na.sup.+ /H.sup.+ antiporter Fafournoux et al. 1991
NADPH-P450 oxidoreductase Tamura et al. 1992
nerve growth factor Buxser et al. 1991
nerve growth factor **receptor** Vissavajjhala et al. 1990
neutrophil NADPH oxidase factors Leto et al. 1991
p47-[phox], p67[phox]
nuclear **hormone receptor** H-2R11BP Marks et al. 1992
nucleolar **protein** p120 Ren et al. 1996
oxytocin **receptor** Gimpl et al. 1995
p53 Patterson et al. 1996
P450 2E1 Patten & Koch 1995
pancreatic lipase Thirstrup et al. 1993
pancreatic procolipase Lowe 1994
papillmoavirus type 11 E1, E2 Bream et al. 1993
papillomavirus type 11-L1 **protein** Rose et al. 1993
papillomavirus type 16 E2 Sanders et al. 1995
papillomavirus type 16 E2 **protein** Sanders et al. 1995
papillomovirus type 16 L1, L2 Xi and Banks, 1991
papillomavirus type 45 L1 major Touze et al. 1996
capsid **protein**
parainfluenza virus type 3, 7, HN, Lehman et al. 1993
7HN
parathyroid **hormone** Mathavan et al. 1995
parvovirus B19 vp1, vp2 Cubie et al. 1993
phospholipase A.sub.2 Abdullah et al. 1995
placental aromatase. . . pre-pro gastrin releasing peptide
Lebacq-verheyden et al. 1988
pro-al(III) chains Tomita et al. 1995
proapoA-I Sorci-Thomas et al. 1996
progesterone **receptor** (A form) Elliston et al. 1992
progesterone **receptors** A&B forms Christensen et al. 1991
prolyl 4-hydroxylase a, b subunits Vuori et al. 1992
prolyl 4-hydroxylae a subunit with. . . George et al. 1996
prostaglandin G/H synthase 1 Barnett et al. 1994
prostaglandin G/H synthase 2 Barnett et al. 1994
protein disulphide isomerase Vuori et al. 1992
protein kinase c-d Rankl et al. 1994
protein kinase Cm Dieterich et al. 1996
pro-urokinase Gao and Hu 1994
rab 6 Yang et al. 1992
rap1A Quilliam et. . . 1996
respiratory syncytial virus F and G Wathen et al. 1989
glycoproteins
retinoblastoma pp110.sup.RB Wang et al. 1990
retinoic acid **receptor** a1 Quick et al. 1994
retinoic acid **receptor** - g1 Reddy et al. 1992
ssDNA-binding **protein** Stigger et al. 1994

sex **steroid**-binding **protein** Sui et al. 1995
(hSBP/hABP, hSHBG)
soluble human insulin **receptor** - Sissom and Ellis 1992
ectodomain
soluble human insulin **receptor** Ahn et al. 1993
tyrosine kinase
Sos1 **protein** Frech et al. 1995
steroid 5a-reductase Iehle et al. 1993
synthetic basic fibroblast growth Hills & Crane-Robinson 1995
factor
TII (CD2) t-lymphocyte surface Richardson et. . . al. 1988
glycoprotein
TII (CD2) Alcover et al. 1988
T-cell leukemia virus type I p40 Nyunoya et al. 1988
T-cell **protein** tyrosine kinase Lehr et al. 1996
T-cell **protein**-tyrosine-phosphatase Zander et al. 1991
T-lymphotropic virus type 1 envelope Yamashita et al. 1992
protein
terminal transferase Chang et al. 1988
terminal deoxynucleotidyl transferase di Primio et al. 1992
thrombomodulin Marumoto et al. 1993
thromboxane synthase Yokoyama et al. 1993
thyroid hormone B.sub.1 **receptor** Putlitz et al.
1991
thyroid peroxidase Kendler et al. 1993
thyrotropin **receptor** extracellular Seetharamaiah et al. 1993
domain
thyrotropin **hormone receptor**- Huang et al. 1993
extracellular domain
tissue inhibitor of metallo- Gomez et al. 1994
proteinases-1
tissue plasminogen activator Jarvis et. . . cell adhesion molecule-1
Stoltenborg et al. 1994
(VCAM1)
vascular endothelial growth factor Fiebich et al. 1993
VEGF.sub.121, VEGF.sub.165
vitamin D **receptor** Nakajima et al. 1993
Vitronectin Zhao and Sane, 1993
Y1 neuropeptide Y **receptor** Munoz et al. 1995
yoked chorionic gonadotropin Narayan et al. 1995
Hyalophora cecropia pupae attacin Gunn et al. 1990
immunodeficiency. . . et al. 1994
human gag and gag-pol Hughes et al. 1993
human gag precursors Royer et al. 1992
human gag-related **proteins** Madisen et al. 1987
human gp120, gp160 Murphy et al. 1990
human gp160, envelope PB1 Dolin et al. 1991
human type 1 envelope glycoproteins Bristow et al. 1994
human type 1 integrase **protein** Rodner et al. 1993
human type 1 matrix **protein** Chazal et al. 1995
human P55 gag Gheysen et al. 1989
human p55 gag and protease Overton et al. 1989
. . . gene, reverse transcriptase Manns and Grosse 1991
and integrase
human type 1 reverse transcriptase Kawa et al. 1993
human tat **protein** Jeang et al. 1988
human type 1 gag precursor Hong and Boulanger 1993
human Type 1 Nef Matsuura et al.. . . haemagglutinin epitope McLinden et
al. 1992
neuraminidase Weyer & Possee, 1991
Price et al. 1989
Mather et al. 1992
polymerase **proteins** St. Angelo et al. 1987
RNA polymerase Kobayashi et al.
RNA polymerase - PB1; PB2; PA Kobayashi et al. 1992
. . . M2 Schroeder et al. 1994

virus recombinant hemagglutinin Treanor et al. 1996
insect:
g-aminobutyric acid (GABA) Shotkoski et al. 1996
receptor
Bombyx mori prothor acicotropic O'Reilly et al. 1995
hormone
ecdysteroid UDP-glucosyl transferase O'Reilly 1995
fungal protease inhibitor F(FPI-F) Pham et al. 1996
juvenile **hormone** esterase Bonning et al. 1995
synthetic pheromone biosynthesis Vakharia et al. 1995
activating neuropeptide
transferrin Winzerling et al. 1996
invertebrate GABA.sub.A **receptor** Smith et al. 1995
b-subunit
JC virus T antigen Bollag et al. 1996
Japanese encephalitis virus:
envelope glycoprotein E Aira 1987
glycoprotein NS1 Flamand et al. 1995
nonstructural **protein** NS1 Flamand et al. 1992
viral structural **proteins** Matsuura et al. 1989
Japanese type hepatitis C virus:
p70(NS3) Hirowatari et al. 1995
p4(NS4A) Hirowatari et al. 1995
p27(NS4B) Hirowatari et al. 1995
p58/56(NS5A) Hirowatari et al. 1995
p66(NS5B) Hirowatari et al. 1995
jellyfish green fluorescet **protein** Eriksson et al. 1996
Johnson grass mosaic virus coat Edwards et al. 1994
protein
juvenile **hormone** esterase Booth et al. 1992
LaCrosse virus:
G1 Pekosz et al. 1995
G2 Pekosz et al. 1995
Lassa virus N **protein** Barber et al. 1990
Lassa virus glycoprotein Hummel et al. 1992
Leishmania virus - capsid Cadd and Patterson 1994
Leishmania. . . locust ion transport peptide Meredith et al. 1996
Louping ill virus - envelope, core and Shiu et al. 1992
membrane **proteins**
luciferase-streptavidin **fusion protein** Karp et al. 1996
lutropin/choriogonadotropin **receptor** Narayan et al. 1996
lymphocyte-specific **protein**-tyrosyl Watts et al. 1992
kinase p56.sup.lck
lymphocytic choriomeningitis virus Min and Bishop 1991
nucleocapsid **protein**
lymphocytic choriomeningitis virus Matsuura et al. 1986
nucleoprotein and glycoprotein
precursor
lymphoid-cell **protein** tyrosine kinase Ramen et al. 1991
p56.sup.lck
maize:
AcTPase Kunze et al. 1995
auxin-binding **protein** Henderson et al. 1995
mitochondrial **protein** URF13 Korth and Levings 1993
transposable element Ac **protein** Hauser et al. 1988
Manduca sexta diuretic **hormone** Maeda 1989
Manduca sexta juvenile **hormone** Touhara et al. 1993
binding **protein**
Marburg virus surface **protein** Becker et al. 1996
Marek's disease virus:
88; 110; 49; 58 Niikura et al. 1992
A antigen Niikura et al. 1991
B antigen Niikura et al. 1992
disease virus type 1 (mDV1)-specific Urakawa et al. 1994
protein p40
virus glycoprotein D Ono et al. 1995

measles virus:
AIK-C strain hemagglutenand **fusion** Takehara et al. 1992
glycoproteins
hemagglutinin Vialard et al. 1990
N **protein** Fooks et al. 1993
nucleoprotein Hummel et al. 1992
melanocortin 1 **receptors** Schioth et al. 1996
mink enteritis parvovirus VP-2 Christensen et al. 1994
minute virus of mice NS-1 Wilson et al.. . . Mokola virus glycoprotein
Tordo et al. 1993
mouse:
CD95/APO1/Fas ligand Mariani et al. 1996
c-fos Corvello et al. 1994
cFos **protein** Corvello et al. 1995
focal adhesion kinase Withers et al. 1996
glutamate **receptor** subunits a1, a2 Kawamoto et al. 1993
growth **hormone** Thordarson et al. 1996
immunophilin FKBP-52 Alnemri et al. 1993
interleukin-3 Hogeland et al. 1992
Knepper et al. 1992
perforin. . . al. 1994
multifunctional mammalian Momoeda et al. 1995
transcription factor, YY1
murine:
a1-subunit of AMPA-selective Kawamoto et al. 1993
glutamate **receptor** channel

L4 ANSWER 2 OF 16 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 2000065645 MEDLINE
DOCUMENT NUMBER: 20065645 PubMed ID: 10598586
TITLE: A fusion protein of the estrogen receptor (ER) and nuclear receptor corepressor (NCoR) strongly inhibits estrogen-dependent responses in breast cancer cells.
AUTHOR: Chien P Y; Ito M; Park Y; Tagami T; Gehm B D; Jameson J L
CORPORATE SOURCE: Division of Endocrinology, Metabolism, and Molecular Medicine, Northwestern University Medical School, Chicago, Illinois 60611, USA.
CONTRACT NUMBER: DK-42144 (NIDDK)
SOURCE: MOLECULAR ENDOCRINOLOGY, (1999 Dec) 13 (12) 2122-36.
Journal code: NGZ; 8801431. ISSN: 0888-8809.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200001
ENTRY DATE: Entered STN: 20000124
Last Updated on STN: 20000124
Entered Medline: 20000110

AB Nuclear **receptor** corepressor (NCoR) mediates repression (silencing) of basal gene transcription by nuclear **receptors** for **thyroid hormone** and retinoic acid. The goal of this study was to create novel estrogen **receptor** (ER) mutants by fusing transferable repressor domains from the N-terminal region of NCoR to a functional ER fragment. Three chimeric NCoR-ER **proteins** were created and shown to lack transcriptional activity. These **fusion proteins** silenced basal transcription of the ERE2-tk-Luc reporter gene and inhibited the activity of co-transfected wild-type ER (wtER), indicating that they possess dominant negative activity. One of the **fusion proteins** (CDE-RD1), containing the ER DNA-binding and ligand-binding domains linked to the NCoR repressor domain (RD1), was selected for detailed examination. Its **hormone** affinity, intracellular localization, and level of expression in transfected cells were similar to wtER, and it bound to the estrogen. . . response element (ERE) DNA in gel shift assays. Glutathione-S-transferase pull-down assays showed that CDE-RD1 retains the

ability to bind to **steroid receptor** coactivator-1. Introduction of a DNA-binding domain mutation into the CDE-RD1 **fusion protein** eliminated silencing and dominant negative activity. Thus, the RD1 repressor domain prevents transcriptional activation despite the apparent ability of CDE-RD1. . . . cancer cells and repressed the growth of T47D cells when delivered to the cells by a retroviral vector. These ER-NCoR **fusion proteins** provide a novel means for inhibiting ER-mediated cellular responses, and analogous strategies could be used to create dominant negative mutants.

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L4 ANSWER 3 OF 16 CAPLUS COPYRIGHT 2001 ACS

ACCESSION NUMBER: 1998:71221 CAPLUS
DOCUMENT NUMBER: 128:136859
TITLE: Soluble ErbB receptor extracellular domain fusion proteins their uses in antagonization of growth factors
INVENTOR(S): Fizpatrick, Vincent Danial; Sliwkowski, Mark; Vandlen, Richard L.
PATENT ASSIGNEE(S): Genentech, Inc., USA
SOURCE: PCT Int. Appl., 58 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9802540	A1	19980122	WO 1997-US11825	19970708
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
CA 2258721	AA	19980122	CA 1997-2258721	19970708
AU 9735962	A1	19980209	AU 1997-35962	19970708
AU 722178	B2	20000727		
EP 912734	A1	19990506	EP 1997-932526	19970708
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
BR 9710357	A	19990817	BR 1997-10357	19970708
JP 2000515372	T2	20001121	JP 1998-506089	19970708

PRIORITY APPLN. INFO.:

US 1996-21640	P	19960712
US 1997-798326	A	19970210
WO 1997-US11825	W	19970708

IT **Steroid receptors**
Thyroid hormone receptors
RL: BPR (Biological process); BUU (Biological use, unclassified); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)
(TR (**thyroid/steroid hormone receptor**), **fusion proteins**; sol. ErbB **receptor** extracellular domain **fusion proteins** their uses in antagonization of growth factors)

DUPLICATE 2

L4 ANSWER 4 OF 16 MEDLINE

ACCESSION NUMBER: 1998101756 MEDLINE
DOCUMENT NUMBER: 98101756 PubMed ID: 9440806
TITLE: TLS (translocated-in-liposarcoma) is a high-affinity interactor for steroid, thyroid hormone, and retinoid

receptors.

AUTHOR: Powers C A; Mathur M; Raaka B M; Ron D; Samuels H H
CORPORATE SOURCE: Division of Molecular Endocrinology, New York University Medical Center 10016, USA.
CONTRACT NUMBER: CA-60945 (NCI)
DK-09211 (NIDDK)
DK-16636 (NIDDK)
+
SOURCE: MOLECULAR ENDOCRINOLOGY, (1998 Jan) 12 (1) 4-18.
Journal code: NGZ; 8801431. ISSN: 0888-8809.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199802
ENTRY DATE: Entered STN: 19980226
Last Updated on STN: 19980226
Entered Medline: 19980218

AB Nuclear **receptors** for **steroid hormones**, **thyroid hormone**, retinoids, and vitamin D are thought to mediate their transcriptional effects in concert with coregulator **proteins** that modulate **receptor** interactions with components of the basal transcription complex. In an effort to identify potential coregulators, **receptor fusions** with glutathione-S-transferase were used to isolate **proteins** in nuclear extracts capable of binding nuclear **hormone receptors**. Glutathione-S-transferase **fusions** with mouse retinoid X **receptor**-alpha enabled the selective isolation of a 65-kDa **protein** (p65) from nuclear extracts of rat and human cells. Binding of p65 to mouse retinoid X **receptor**-alpha was centered around the DNA-binding domain. p65 also bound regions encompassing the DNA-binding domain in estrogen, **thyroid hormone**, and glucocorticoid **receptors**. p65 was identified as TLS (translocated-in-liposarcoma), a recently identified member of the RNP family of nuclear RNA-binding **proteins** whose members are thought to function in RNA processing. The N-terminal half of TLS bound to **thyroid hormone receptor** with high affinity while the **receptor** was bound to appropriate DNA target sites. Functional studies indicated that the N-terminal half of TLS can interact with **thyroid hormone receptor** in vivo. TLS was originally discovered as part of a **fusion protein** arising from a chromosomal translocation causing human myxoid liposarcomas. TLS contains a potent transactivation domain whose translocation-induced **fusion** with a DNA-binding **protein** (CHOP) yields a powerful transforming oncogene and transcription factor. The transactivation and RNA-binding properties of TLS and the nature of its interaction with nuclear **receptors** suggest a novel role in nuclear **receptor** function.

L4 ANSWER 5 OF 16 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 97271687 MEDLINE
DOCUMENT NUMBER: 97271687 PubMed ID: 9126603
TITLE: One-step immunoaffinity purification of recombinant human retinoic acid receptor gamma.
AUTHOR: Repa J J; Berg J A; Kaiser M E; Hanson K K; Strugnell S A; Clagett-Dame M
CORPORATE SOURCE: Interdepartmental Graduate Program in Nutritional Sciences,
University of Wisconsin-Madison 53706, USA.
CONTRACT NUMBER: DK-14881 (NIDDK)
SOURCE: PROTEIN EXPRESSION AND PURIFICATION, (1997 Apr) 9 (3) 319-30.
Journal code: BJV; 9101496. ISSN: 1046-5928.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199705
ENTRY DATE: Entered STN: 19970602
Last Updated on STN: 19970602
Entered Medline: 19970519

AB Retinoic acid **receptors** (RAR) are members of the **steroid /thyroid hormone receptor** superfamily and serve as ligand-activated transcription factors. In order to facilitate studies of **receptor protein**, we have generated a monoclonal antibody to the human RAR gamma, and have developed a procedure to purify the full-length **receptor** expressed in insect cells. The monoclonal antibody (A10) was developed using as antigen a carboxy-terminal fragment of the human RAR gamma expressed as a bacterial **fusion protein**. The A10 monoclonal antibody binds to both native and denatured forms of the human RAR gamma. This antibody was immobilized on a resin and used to purify full-length, baculovirus-expressed human RAR gamma to near homogeneity. The immunoaffinity-purified **receptor** is > 90-95% pure as revealed by silver-stained gels. The identity of the single **protein** band as RAR gamma was verified by immunoblotting using a polyclonal antibody to an epitope distinct from that recognized by. . . retinoic acid response element was also studied. Response element binding by RAR gamma required the presence of the retinoid X **receptor**, but did not require the presence of additional **proteins**. Human RAR gamma **protein** purified in this fashion will be useful in future structural and functional studies.

L4 ANSWER 6 OF 16 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 1998057341 MEDLINE
DOCUMENT NUMBER: 98057341 PubMed ID: 9396634
TITLE: Transfection of TTF-1 gene induces thyroglobulin gene expression in undifferentiated FRT cells.
AUTHOR: Mascia A; De Felice M; Lipardi C; Gentile R; Cali G; Zannini M; Di Lauro R; Nitsch L
CORPORATE SOURCE: Centro di Endocrinologia ed Oncologia Sperimentale del CNR - Dipartimento di Biologia e Patologia Cellulare e Molecolare, Universita degli Studi di Napoli Federico II, Naples, Italy.
SOURCE: BIOCHIMICA ET BIOPHYSICA ACTA, (1997 Nov 1) 1354 (2) 171-81.
Journal code: A0W; 0217513. ISSN: 0006-3002.
PUB. COUNTRY: Netherlands
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199801
ENTRY DATE: Entered STN: 19980122
Last Updated on STN: 19980122
Entered Medline: 19980105

AB The thyroglobulin gene, the substrate for **thyroid hormone** biosynthesis, is not expressed in the FRT cell line, which, even though it manifests the polarised epithelial phenotype, does not express any of the **thyroid** functional properties. Two transcription factors, TTF-1 and Pax-8, have been implicated in **thyroid** specific expression of the thyroglobulin gene. FRT cells contain Pax-8 but they lack TTF-1. In this paper, we show that. . . expression vectors in FRT cells results in activation of thyroglobulin gene expression. If the expression vector encoded for TTF-1-ER, a **fusion** gene coding for the entire TTF-1 **protein** fused to the **hormone**-binding domain of the **steroid receptor**, under the control of the RSV promoter, thyroglobulin gene expression was controlled by estrogen. These data provide a direct demonstration that TTF-1 activates the chromosomal thyroglobulin promoter.

Since transfection of TTF-1 expression vectors in non-**thyroid** cell types did not result in thyroglobulin gene expression, it is suggested that Pax-8, in addition, perhaps, to a specific cellular environment, might be required for **thyroid** specific expression of the thyroglobulin gene.

L4 ANSWER 7 OF 16 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1996:388480 CAPLUS
DOCUMENT NUMBER: 125:105138
TITLE: A novel steroid hormone receptor and compounds that activate it for potentiation of G-protein-coupled receptors
INVENTOR(S): Friedman, Eitan; Holloway, M. Katharine; Rodan, Gideon
A.; Schmidt, Azriel; Vogel, Robert L.
PATENT ASSIGNEE(S): Merck and Co., Inc., USA; Medical College of Pennsylvania
SOURCE: PCT Int. Appl., 62 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9613257	A1	19960509	WO 1995-US13931	19951024
W: AL, AM, AU, BB, BG, BR, BY, CA, CN, CZ, EE, FI, GE, HU, IS, JP, KG, KR, KZ, LK, LR, LT, LV, MD, MG, MK, MN, MX, NO, NZ, PL, RO, RU, SG, SI, SK, TJ, TM, TT, UA, US, UZ				
RW: KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
US 5607967	A	19970304	US 1994-330518	19941027
CA 2200886	AA	19960509	CA 1995-2200886	19951024
AU 9539700	A1	19960523	AU 1995-39700	19951024
AU 705987	B2	19990603		
EP 786995	A1	19970806	EP 1995-937658	19951024
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, NL, PT, SE				
JP 10509139	T2	19980908	JP 1995-514767	19951024
PRIORITY APPLN. INFO.:			US 1994-330518	19941027
			WO 1995-US13931	19951024

IT **Thyroid hormone receptors**
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(**fusion** products with NER **receptor**, for identification of NER ligands; novel **steroid hormone receptor** and compds. that activate it for potentiation of G-**protein**-coupled **receptors**)

IT **Receptors**
RL: BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(**thyroid hormone**, **fusion** products with NER **receptor**, for identification of NER ligands; novel **steroid hormone receptor** and compds. that activate it for potentiation of G-**protein**-coupled **receptors**)

L4 ANSWER 8 OF 16 USPATFULL
ACCESSION NUMBER: 96:108853 USPATFULL
TITLE: Receptor transcription-repression activity compositions
and methods
INVENTOR(S): Evans, Ronald M., La Jolla, CA, United States
Hollenberg, Stanley M., Seattle, WA, United States
Oro, Anthony E., San Diego, CA, United States

PATENT ASSIGNEE(S): The Salk Institute for Biological Studies, La Jolla, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5578483		19961126
APPLICATION INFO.:	US 1991-691043		19910621 (7)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1988-289561, filed on 23 Dec 1988, now abandoned

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Wax, Robert A.
ASSISTANT EXAMINER: Carlson, K. Cochrane
LEGAL REPRESENTATIVE: Pretty, Schroeder, Brueggemann & Clark, Reiter, Stephen E., Ramos, Robert T.
NUMBER OF CLAIMS: 8
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 5 Drawing Figure(s); 4 Drawing Page(s)
LINE COUNT: 1064
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DRWD FIG. 4 represents the results of experiments on trans-repression and trans-activation activities of carboxy-terminal mutants of hGR. Such mutants include **fusion proteins**, with the carboxy-terminus of the wild-type hGR (by which is intended the part of the primary sequence of the **receptor** from amino acid 487 and higher, i.e., the part, including the ligand-binding domain, carboxy-terminal of the DNA-binding domain) replaced by. . . for FIGS. 2 and 3, except that the RSV plasmid used in controls 1 and 2 in place of the **receptor**- or **receptor**-analog-expressing plasmid had the **thyroid hormone receptor** -encoding cDNA inserted in the anti-sense orientation downstream of the RSV promoter rather than the beta-galactosidase-encoding DNA in the sense orientation. . . GGM consists of amino acids 1-489 of hGR as the N-terminal part and amino acids 671-984 of hMR (human mineralocorticoid **receptor**) as the C-terminal part. Mutant GGM was made from a cDNA constructed by first introducing an additional XhoI site into. . . XhoI site of the hGR-encoding sequence. With the wild-type hGR, control 1, and the three mutants other than GGM, the **steroid** used was dexamethasone. With the mutant with beta-gal at the carboxy-terminus, normalization for transfection efficiency was based on data from. . . described for FIG. 1 supra, in which beta-galactosidase is expressed from the RSV promoter. With GGM and Control 2, the **steroid** used was aldosterone. Control 2 was the same as Control 1 except for the substitution of aldosterone for dexamethasone.

L4 ANSWER 9 OF 16 USPATFULL

ACCESSION NUMBER: 95:49945 USPATFULL
TITLE: Heterovesicular liposomes
INVENTOR(S): Kim, Sinil, Solana Beach, CA, United States
PATENT ASSIGNEE(S): DepoTech Corporation, La Jolla, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5422120		19950606
APPLICATION INFO.:	US 1993-78701		19930616 (8)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1988-196590, filed on 30 May 1988, now abandoned which is a continuation-in-part of Ser. No. US 1990-496846, filed on 21 Mar 1990, now abandoned

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted

PRIMARY EXAMINER: Kishore, Gollamudi S.
LEGAL REPRESENTATIVE: Spensley Horn Jubas & Lubitz
NUMBER OF CLAIMS: 46
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 8 Drawing Figure(s); 1 Drawing Page(s)
LINE COUNT: 925
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
DETD TABLE 1

Antiasthmatics
Antiarrhythmics
Tranquilizers
melairoterenol
propanolol chlorpromazine
aminophylline atenolol benzodiazepine
theophylline verapamil butyrophenones
terbutaline Antianginas
hydroxyzines
norepinephrine
isosorbide dinitrate
meprobamate
ephedrine **Hormones** phenothiazines
isoproterenol thyroxine thioxanthenes
adrenalin corticosteroids
Steroids
Cardiac glycosides
testosterone
preunisone
digitalis estrogen triamcinolone
digitoxin progesterone
hydrocortisone
lanatoside C mineralocorticoid
dexamethasone
digoxin Antidiabetics
betamethasone
Antihypertensives
Diabenese preunisolone
apresoline insulin Antihistamines
atenolol Antineoplastics
pyribenzamine
captopril azathioprine
chlorpheniramine
reserpine. . . the treatment of allergies
influenza
respiratory syncytial virus
HIV vaccine
Hemophilus influenza vaccines
Hepatitis A,B,C vaccines
mumps
rubella
measles
tetanus
malaria vaccines
herpes
cancer vaccines
Anti-leu-3a vaccine
Monoclonal Antibodies (human, mouse other species-derived and/or recombinant
and/or **fusions** and/or fragments thereof)
OKT3
OKT4
HA-IA
Anti-Carcino-Embryonic Antigen Antibodies
Anti-Ganglioside Antibodies: Anti GD2, Anti GM2, Anti GD3, Anti GM3
Urinary Tract-Associated Antigen-related antibodies
Anti-Il-2 **Receptor**

Chimeric Anti-Leu-2
Anti-IL-2 **receptor**
Anti-Leu-2
Chimeric Anti-Leu-3a
Chimeric L6
MAb-L6
Radiolabeled L6
Centorex
Centoxin
Panorex
Anti-LPS
Immunotoxin
Anti-tumor necrosis factor
Anti-pseudomonas
Anti-tumor necrosis factor
OncoRad 103
OncoScint CR103
OncoScint OV103
OncoScint PR356
OncoTher 130
KS 1/4-DAVLB
ADCC agent
Murine monoclonal antibodies to human B-cell lymphomas. . . isooctyl ester),
2,4,5-T amine (2,4,5-trichlorophenoxyacetic acid trimethylamine)
other triazine herbicides
other chloroacetamide herbicides
other phenoxyacid herbicides
Pesticides
Abamectin
other avermectins
atrazine
lindane
dichlorvos
dimethoate
warfarin
p,p'-DDD
p,p'-DDE
HCH
DMDT
aldrin
dieldrin
Aldicarb
EDB
DCP
DBCP
simazine
cyanazine
Bacillus thuringiensis toxin
Bacillus thuringiensis var. kurstaki
bis(tri-n-butyltin)oxide (TBTO)
other organochlorine pesticides
Proteins and Glycoproteins
lymphokines
interleukins - 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11.
cytokines
GM-CSF
M-CSF
G-CSF
tumor necrosis factor
inhibin
tumor growth factor
Mullerian inhibitors substance
nerve growth factor
fibroblast growth factor
platelet derived growth factor

coagulation factors (e.g. VIII, IX, VII)
insulin
tissue plasminogen activator
histocompatibility antigen
oncogene products
myelin basic **protein**
collagen
fibronectin
laminin
other **proteins** made by recombinant DNA technology
erythropoietin
IL-3/GM-CSF **fusion proteins**
Monoclonal antibodies
Polyclonal antibodies
antibody-toxin **fusion proteins**
antibody radionuclide conjugate
Interferons
Fragments and peptide analogs, and analogs of fragment of **proteins**, peptides
and glycoproteins.
Epidermal growth factor
CD4 **receptor** and other recombinant **receptors**
other **proteins** isolated from nature
Antidiuretic **hormone**
oxytocin
adrenocorticotropin **Hormone**
calcitonin
follicle stimulating **hormone**
luteinizing **hormone** releasing **hormone**
luteinizing **hormone**
gonadotrophin
transforming growth factors
streptokinase
Human Growth **Hormone**,
Somatotropins for other species, including, but not limited to:
1. Porcine,
2. Bovine,
3. Chicken,
4. Sheep,
5. Fish,

Growth **Hormone** releasing **hormones** for humans and various animal species,
Glucagon,
Desmopressin,
Thyroid Releasing **Hormone**,
Thyroid Hormone,
Secretin,
Magainins,
Integrins,
Adhesion Peptides, including, but not limited to, those having the Arginine-Glutamine-Aspartic Acid sequence,
Super Oxide Dismutase,
Defensins,
T-Cell **Receptors**,
Bradykinin antagonists,
Pentigetide,
Peptide T,
Antinflammins,
Major Histocompatibility (MHC) complex components and peptides targeted to the MHC,
Protease inhibitors,
Lypressin,
Buserelin,
Leuprolide,
Nafarelin,
Deslorelin,

Goserelin,
Historelin,
Triptorelin,
LHRH antagonists,
HOE-2013,
Detirelix,
Org-30850,
ORF-21243,
Angiotensin Converting Enzyme inhibitor Peptide,
Renin inhibitory peptides,
Ebiratide (HOE-427),
DGAVP,
Opiate **receptor** agonists and antagonists, including, but not limited to:
1. Enkephalins,
2. Endorphins,
E-2078,
DPDPE,
Vasoactive intestinal peptide,
Atrial Natriuretic Peptide,
Brain Natriuretic Peptide,
Atrial Peptide clearance inhibitors,
Hirudin,
Oncogene Inhibitors,
Other Colony Stimulating Factors,
Neurotransmitters

	Radionuclides	Radio contrasts
Dopamine	Technetium	Gadolinium chelates
Epinephrine	Indium	Iohexol
Norepinephrine		
	Yttrium	Ethiodol
acetylcholine	Gallium	Iodexinol

Gammaamino butyric acid
Others
amino acids
vitamins
cell surface **receptor** blockers

L4 ANSWER 10 OF 16 MEDLINE DUPLICATE 5

ACCESSION NUMBER: 96102134 MEDLINE
DOCUMENT NUMBER: 96102134 PubMed ID: 8524784
TITLE: A protein that interacts with members of the nuclear hormone receptor family: identification and cDNA cloning.
AUTHOR: Zeiner M; Gehring U
CORPORATE SOURCE: Institut fur Biologische Chemie, Universitat Heidelberg, Germany.
SOURCE: PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (1995 Dec 5) 92 (25) 11465-9. Journal code: PV3; 7505876. ISSN: 0027-8424.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-Z35491
ENTRY MONTH: 199601
ENTRY DATE: Entered STN: 19960219
Last Updated on STN: 19960219
Entered Medline: 19960124

AB In search of **proteins** which interact with activated **steroid hormone receptors**, we screened a human liver lambda gt11 expression library with the glucocorticoid **receptor**. We identified and cloned a cDNA sequence of 1322 bp that encodes a **protein** of 274 aa. This **protein** consists predominantly of hydrophilic amino acids and contains a putative bipartite

nuclear localization signal. The in vitro translated **receptor** -associating **protein** runs in SDS/polyacrylamide gels with an apparent molecular mass of 46 kDa. By use of the bacterially expressed **fusion protein** with glutathione S-transferase we have found that interaction is not limited to the glucocorticoid **receptor** but included other nuclear **receptors**--most notably, the estrogen and **thyroid receptors**. Binding also occurs with the glucocorticoid **receptor** complexed with the antiglucocorticoid RU 38486, with the estrogen **receptor** complexed with the antiestrogen 4-hydroxytamoxifen or ICI 164,384, and even with **receptors** not complexed with ligand. Association with **steroid hormone receptors** depends on prior **receptor** activation--i.e., release from heat shock **proteins**. The sequence identified here appears to be a general partner **protein** for nuclear **hormone receptors** , with the gene being expressed in a variety of mammalian tissues.

L4 ANSWER 11 OF 16 MEDLINE DUPLICATE 6

ACCESSION NUMBER: 95349617 MEDLINE
DOCUMENT NUMBER: 95349617 PubMed ID: 7623841
TITLE: A 10-amino-acid sequence in the N-terminal A/B domain of thyroid hormone receptor alpha is essential for transcriptional activation and interaction with the general transcription factor TFIIB.
AUTHOR: Hadzic E; Desai-Yajnik V; Helmer E; Guo S; Wu S; Koudinova N; Casanova J; Raaka B M; Samuels H H
CORPORATE SOURCE: Department of Cell Biology, New York University Medical Center, New York 10016, USA.
CONTRACT NUMBER: 5T35DK07421 (NIDDK)
DK16636 (NIDDK)
GM07238 (NIGMS)
+
SOURCE: MOLECULAR AND CELLULAR BIOLOGY, (1995 Aug) 15 (8) 4507-17. Journal code: NGY; 8109087. ISSN: 0270-7306.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199508
ENTRY DATE: Entered STN: 19950911
Last Updated on STN: 19950911
Entered Medline: 19950829

AB The effects of the **thyroid hormone** (3,5,3'-triiodo-L-thyronine [T3]) on gene transcription are mediated by nuclear T3 **receptors** (T3Rs). alpha- and beta-isoform T3Rs (T3R alpha and -beta) are expressed from different genes and are members of a superfamily of ligand-dependent transcription factors that also includes the **receptors** for **steroid hormones**, vitamin D, and retinoids. Although T3 activates transcription by mediating a conformational change in the C-terminal approximately 220-amino-acid ligand-binding domain. . . of the 50-amino-acid N-terminal A/B domain of chicken T3R alpha (cT3R alpha) decreases T3-dependent stimulation of genes regulated by native **thyroid hormone** response elements about 10- to 20-fold. The requirement of the A/B region for transcriptional activation was mapped to amino acids. . . amino acids. The A/B region of cT3R alpha is not required for T3 binding or for DNA binding of the **receptor** as a heterodimer with retinoid X **receptor**. In vitro binding studies indicate that the N-terminal region of cT3R alpha interacts efficiently with TFIIB and that this interaction. . . in the binding of cT3R alpha includes an amphipathic alpha helix contained within residues 178 to 201. Analysis using a **fusion protein** containing the DNA-binding domain of GAL4 and the entire A/B region of cT3R alpha suggests that this region does not. . .

L4 ANSWER 12 OF 16 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1994:316754 CAPLUS
DOCUMENT NUMBER: 120:316754
TITLE: A cDNA for a novel member of the steroid/thyroid hormone receptor family
INVENTOR(S): Kroczek, Richard; Mages, Hans Werner
PATENT ASSIGNEE(S): Germany
SOURCE: PCT Int. Appl., 33 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9404675	A2	19940303	WO 1993-EP2223	19930819
WO 9404675	A3	19941208		

W: AU, BB, BG, BR, CA, CZ, FI, HU, JP, KP, KR, KZ, LK, MG, MN, MW, NO, NZ, PL, RO, RU, SD, SK, UA, US, VN
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: EP 1992-114134 19920819

IT **Receptors**
RL: BIOL (Biological study)
(TR (**thyroid/steroid hormone receptor**), nuclear **receptor** of T-cells (NOT), cDNA for, cloning and expression of, manuf. of **fusion proteins** in relation to)

L4 ANSWER 13 OF 16 MEDLINE DUPLICATE 7
ACCESSION NUMBER: 95057285 MEDLINE
DOCUMENT NUMBER: 95057285 PubMed ID: 7967725
TITLE: The retinoid receptors.
AUTHOR: Pemrick S M; Lucas D A; Grippo J F
CORPORATE SOURCE: Department of Toxicology and Pathology, Hoffman-La Roche, Nutley, NJ 07110.
SOURCE: LEUKEMIA, (1994 Nov) 8 (11) 1797-806. Ref: 114
Journal code: LEU; 8704895. ISSN: 0887-6924.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, ACADEMIC)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199412
ENTRY DATE: Entered STN: 19950110
Last Updated on STN: 19970203
Entered Medline: 19941213

AB The retinoid **receptors** belong to a large superfamily of ligand-inducible transcription factors that include the **steroid**, vitamin D and **thyroid hormone receptors**, the peroxisome proliferator-activated **receptor**, the insect edysteroid **receptor**, and a number of orphan **receptors** whose ligands are unknown. All nuclear **receptors** have several well-characterized structural domains, including a conserved DNA-binding domain, and a ligand binding domain at the carboxyl terminus of the **receptor**. The RAR and RXR classes of nuclear retinoic acid **receptors** are each composed of alpha, beta and gamma subtypes with more than one isoform for each **receptor** subtype. Data from many investigators suggest there are RAR- and RXR-dependent gene pathways, and that the individual **receptor** subtypes may control distinct gene expression patterns. In addition, RXR has been found to heterodimerize with other nuclear **receptors** to form active transcriptional complexes, which influence the activity of a variety of gene pathways important in growth and differentiation.. . . In the latter case,

retinoid resistance has been associated with a mutation in the RAR gene which transcribes a RAR **receptor** truncated at the C-terminal end. These mutated RAR **receptors** exhibit a reduced affinity for retinoic acid while retaining the ability to bind to a retinoic acid response element on DNA. As a result, these mutant **receptors** exhibit dominant-negative activity by binding to the DNA without activating transcription and by competing with other **receptors** for sites on the response element. In fact, dominant-negative activity may be very important in the development of many neoplastic. . . promyelocytic leukemia (APL), where a t(15;17) chromosomal translocation fuses the PML gene to the RAR gene, to produce a PML-RAR **fusion protein** in large excess in the cell. However, retinoid resistance in the patient is most probably the result of pharmacokinetic problems,. . .

L4 ANSWER 14 OF 16 MEDLINE DUPLICATE 8

ACCESSION NUMBER: 95106823 MEDLINE
DOCUMENT NUMBER: 95106823 PubMed ID: 7808017
TITLE: The retinoid receptors.
AUTHOR: Pemrick S M; Lucas D A; Grippo J F
CORPORATE SOURCE: Department of Toxicology and Pathology, Hoffmann-La Roche, Nutley, NJ 07110.
SOURCE: LEUKEMIA, (1994) 8 Suppl 3 S1-10. Ref: 114
Journal code: LEU; 8704895. ISSN: 0887-6924.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, ACADEMIC)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199502
ENTRY DATE: Entered STN: 19950215
Last Updated on STN: 19950215
Entered Medline: 19950202

AB The retinoid **receptors** belong to a large superfamily of ligand-inducible transcription factors that include the **steroid**, vitamin D and **thyroid hormone receptors**, the peroxisome proliferator-activated **receptor**, the insect edysteroid **receptor**, and a number of orphan **receptors** whose ligands are unknown. All nuclear **receptors** have several well-characterized structural domains, including a conserved DNA-binding domain, and a ligand binding domain at the carboxyl terminus of the **receptor**. The RAR and RXR classes of nuclear retinoic acid **receptors** are each composed of alpha, beta and gamma subtypes with more than one isoform for each **receptor** subtype. Data from many investigators suggest there are RAR- and RXR-dependent gene pathways, and that the individual **receptor** subtypes may control distinct gene expression patterns. In addition, RXR has been found to heterodimerize with other nuclear **receptors** to form active transcriptional complexes, which influence the activity of a variety of gene pathways important in growth and differentiation.. . . In the latter case, retinoid resistance has been associated with a mutation in the RAR gene which transcribes a RAR **receptor** truncated at the C-terminal end. These mutated RAR **receptors** exhibit a reduced affinity for retinoic acid while retaining the ability to bind to a retinoic acid response element on DNA. As a result, these mutant **receptors** exhibit dominant-negative activity by binding to the DNA without activating transcription and by competing with other **receptors** for sites on the response element. In fact, dominant-negative activity may be very important in the development of many neoplastic. . . promyelocytic leukemia (APL), where a t(15;17) chromosomal translocation fuses the PML gene to the RAR gene, to produce a PML-RAR **fusion protein** in large excess in the cell. However, retinoid resistance in the patient is most probably the result of pharmacokinetic problems,.

L4 ANSWER 15 OF 16 MEDLINE DUPLICATE 9

ACCESSION NUMBER: 93275337 MEDLINE
DOCUMENT NUMBER: 93275337 PubMed ID: 8389001
TITLE: The retinoic acid receptor-beta 2 contains two separate cell-specific transactivation domains, at the N-terminus and in the ligand-binding domain.
AUTHOR: Folkers G E; van der Leede B J; van der Saag P T
CORPORATE SOURCE: Hubrecht Laboratory, Netherlands Institute for Developmental Biology, Utrecht.
SOURCE: MOLECULAR ENDOCRINOLOGY, (1993 Apr) 7 (4) 616-27.
Journal code: NGZ; 8801431. ISSN: 0888-8809.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199306
ENTRY DATE: Entered STN: 19930716
Last Updated on STN: 19980206
Entered Medline: 19930629

AB In contrast to other members of the **steroid/thyroid hormone** superfamily, not much is known about the regions involved in transactivation of the **receptors** for retinoic acid. To determine the transactivation function of RARs, **fusion proteins** between the DNA-binding domain of the yeast transcription factor GAL4 and retinoic acid **receptor**-alpha (RAR alpha) or RAR beta were made. Transfection of these constructs resulted in RA-induced activation of a GAL4-responsive element-containing promoter.. . . function. Internal deletions in the ligand-binding domain in both GAL-RAR beta and RAR beta expression constructs resulted in a nonfunctional **receptor**, indicating that the complete ligand-binding domain is required for its transactivation function. Furthermore, we have shown that the contribution of. . .

L4 ANSWER 16 OF 16 MEDLINE DUPLICATE 10

ACCESSION NUMBER: 91061753 MEDLINE
DOCUMENT NUMBER: 91061753 PubMed ID: 2247065
TITLE: The NGFI-B protein, an inducible member of the thyroid/steroid receptor family, is rapidly modified posttranslationally.
AUTHOR: Fahrner T J; Carroll S L; Milbrandt J
CORPORATE SOURCE: Department of Pathology, Washington University School of Medicine, St. Louis, Missouri 63110.
CONTRACT NUMBER: NS01018 (NINDS)
PO1 CA49712 (NCI)
SOURCE: MOLECULAR AND CELLULAR BIOLOGY, (1990 Dec) 10 (12) 6454-9.
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AB . . . NGFI-B gene is rapidly activated by a variety of stimuli that induce cells to differentiate or proliferate. It encodes a **protein** with a predicted molecular mass of congruent to 61 kDa and is a member of the **thyroid/steroid hormone receptor** gene family. To characterize this **protein**, monoclonal antibodies were raised against a bacterial TrpE-NGFI-B **fusion protein** that encompasses a large portion (Glu-410 to Leu-527) of the carboxy-terminal domain of NGFI-B. These antibodies detected a **protein** that was rapidly synthesized in response to nerve growth

factor (NGF) and migrated as a broad band on sodium dodecyl. . . that ranged from 63 to 88 kDa. Pulse-chase analysis demonstrated that NGFI-B was rapidly posttranslationally modified and was a short-lived **protein**. NGFI-B was found to be a phosphorylated **protein**, and the multiple NGFI-B species coalesced into a single, more rapidly migrating species when treated with alkaline phosphatase. PC12 cells. . .

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